Exhibit A

[redacted]

Particularly when viewed in the context of the advances in the mAb therapy field in general, the attributes and strengths of mAbs are particularly well-suited to the demands of prostate cancer therapy:

1. mAbs can specifically localize to disseminated tumor sites at levels orders of magnitude higher than normal tissues.

2. Therapeutic efficacy has been proven in tumor types (e.g., colon cancer and NHL) where the clinical setting resembles prostate cancer.

3. mAbs have a number of potential mechanisms of anti-tumor activity including:

    a. the relative radiosensitivity of PCa provides one potential class of cytotoxic agents to specifically deliver to tumor sites by way of mAb.

    b. mAbs can trigger the host's own immune response to tumor.

4. Prostate cancer metastases are small-volume sites (typically measured in microns or mm) ideal for radioisotope or immunotherapy.

5. The availability of established parameters such as PSA and pathological features (e.g., stage, Gleason score, seminal vesicle invasion, positive margins, nodal disease, etc.), provide appropriate indications for adjuvant mAb therapy where such therapy is likely to be most beneficial.

6. Last, but not least, is the fact that mAbs are non-toxic.

[redacted]

We believe that we are well on the way to prove that these advantages are more than just theoretical. We have recently completed our mAb Prost 30 biodistribution study in 15 patients with prostate cancer. Doses ranged from 1.0 to 20.0 mg of mAb. Fourteen of the 15 patients had their prostates *in situ* and were evaluable for localization of Prost 30. In all 14 of these cases, including two with prior radiation therapy, the prostate was successfully imaged. In two cases, patients had known sites of metastatic disease imaged on conventional CT scans: regional lymph nodes (both patients) and liver (1 patient). In these cases, these sites also were successfully imaged with Prost 30. In four cases, after resecting the prostate one week after mAb administration, the prostate specimens were scanned alongside specimens of blood drawn at the time of the resection (see appended representative figure). These studies confirmed specific uptake in the prostate at substantially higher levels than in the blood, and this uptake persists for more than one week. No patient on the trial had any side effects.

Exhibit A (cont)

[redacted]

Having established the ability of the mAb to localize to disseminated sites of disease, more interesting and potentially far more important is the observation that two hormone-refractory patients with progressively rising PSAs prior to entry on the imaging trial responded with substantial (75%) decreases in their PSAs each lasting 10 months after a single 5 mg dose of Prost 30. None of the other patients on the trial are evaluable for response due to receiving other therapy in addition to Prost 30. As the isotope dose used in this biodistribution trial was too low to explain the responses, we believe the responses were due to the mAb triggering an endogenous anti-tumor immune response. Another interesting and provocative observation is that this trial included 6 "high-risk" patients (high PSA ± high Gleason ± high stage) who underwent radical prostatectomy plus Prost 30 treatment. None of these patients have demonstrated signs of relapse (either metastatic disease or (PSA) failure) with a median follow-up of almost 2 years.

[redacted] unconjugated ("naked") Prost 30 in a series of patients evaluable for response. This was to establish the safety of naked antibody-as a prelude to an adjuvant trial similar to that already shown effective in colon cancer -- and to provide a benchmark with which to compare the results of a radiolabeled mAb trial. Doses range from 1.25 mg to 5.0 mg -- the level at which we saw the responses in the earlier trial. Fifteen weeks into this trial we have entered 16 patients. Many of the patients, including some at the lowest dose level, have responded with declines in PSA ranging from 25-55%. It is obviously too early to discuss duration of response. While this data is exceedingly preliminary, it is certainly provocative, particularly as the mAb has no conjugated cytotoxic moiety.

We have also developed in the laboratory a higher affinity Prost 30 mAb which we have designated Prost 130. Prost 130 binds the same antigen as Prost 30, but at a different, and repeated, site. It is possible that these mAbs (Prost 30 and 130) will be additive or synergistic in combination. Two other mAbs we have recently developed, C37 and C219, have demonstrated both prostate specificity (in vitro and in vivo) and the ability to directly lyse LNCaP cells in vitro in the presence of human serum as a source of complement. Furthermore, the cytotoxicity of these mAbs are synergistic when combined in vitro. [redacted] these three mAbs (Prost 130, C37 and C219) have been contracted to an FDA-approved manufacturer for production of clinical grade material for upcoming trials.

[redacted]